

Giant T Wave Inversion

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The genesis of the T wave of the electrocardiogram is ill understood. It represents ventricular repolarization, and is thus dependent on changes occurring at cell membrane level. It is the most labile component of the QRST complex and is affected by many physiological, neuro-humoral, and physical factors in health, as well as being the most sensitive index of disease.

One of the most interesting and least understood deformities is that of massive enlargement of the T wave, in which the T wave is grotesquely blown up, often inverted, and covers an area many times that of the QRS complex. Coronary artery disease has often been implicated, especially when the T waves are deeply inverted (Garcia-Palmieri *et al.*, 1956; Ippolito, Blier, and Fox, 1954; Silverman and Goodman, 1949; Pruitt, Klakeg, and Chapin, 1955; Wood and Wolferth, 1934; Fisch, 1961; Costanzo, de la Pierre, and Pietra, 1962), but coronary disease is probably not basically responsible when the deformity is gross. Ippolito *et al.* (1954) first drew attention to the occurrence of massive T wave inversion with complete heart block, and this observation has been confirmed by others (Pfeferman, Chansky, and Fehér, 1960; Szilagyi and Solomon, 1959; Jedlička and Panoš, 1962; Birke and Ström, 1955; Holzmänn, 1960; Zoob and Smith, 1963).

CAUSES OF GIANT T WAVE INVERSION

During the past few years we have encountered massive T wave inversion in a variety of conditions. It would be worth while to review the causes of giant T wave inversion and to stress the features of difference or similarity in the following conditions:

(1) Stokes-Adams attacks associated with complete heart block; (2) ischaemic heart disease (coronary artery disease); (3) bradycardia; (4) right

ventricular hypertrophy and right bundle-branch block; (5) metabolic disturbances; (6) changes during coronary angiography; and (7) cerebral disturbances.

(1) *Giant T Wave Inversion with Complete Heart Block.* Deep, obtuse, negative, asymmetrical, broad waves have been encountered in patients with Stokes-Adams attacks and loss of consciousness. We believe that this is a specific electrocardiographic syndrome unassociated with coronary disease, and that several factors are involved in its causation. Examples of this type of deformity are shown in Fig. 1. We have encountered 7 patients with this disturbance following Stokes-Adams attacks, due to complete heart block, and one with complete heart block, without an overt syncopal attack. The T waves are characteristically massive, distorted, and strikingly asymmetrical. They are best seen in the chest leads V2 to V4 and are unstable, though the changes often persist for several days before disappearing. In the few publications on this syndrome that we have been able to find, only Holzmänn (1960) seems to have been aware of this unique electrocardiographic pattern as a definite entity, which is labelled "das postsynkopale Brady-cardie-Stoffwechsel Syndrom". Lenègre and Moreau (1962) suggested that this T wave pattern after a Stokes-Adams attack indicated specifically that ventricular fibrillation, and not standstill, was responsible for the attack. Two well-documented cases were recorded by Birke and Ström (1955), and Soscia, Fusco, and Grace (1964). The mechanism of syncope in 2 of our patients was seen to be ventricular fibrillation, but this must be interpreted with caution because so often both standstill and fibrillation coexist in the same patient. We believe that cerebral reflexes are mainly responsible for those changes (Jacobson and Schrire, 1965).

Received November 15, 1965.

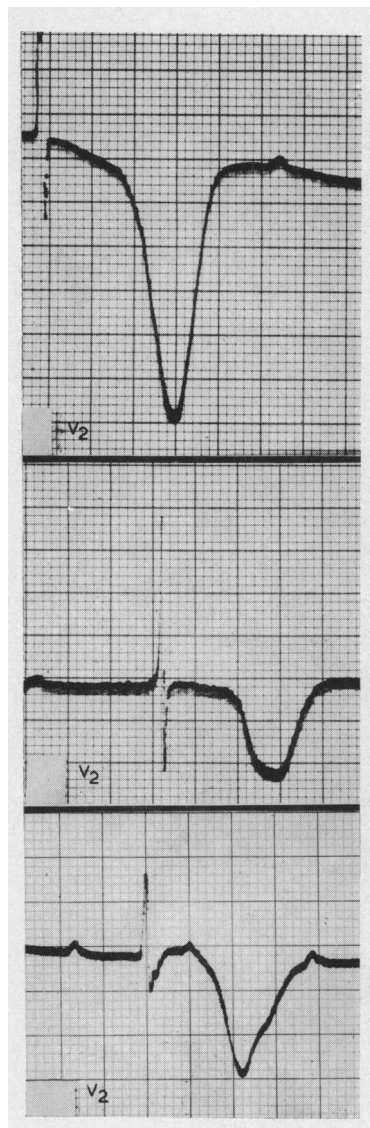


FIG. 1.—Giant T wave inversion in 3 patients with complete heart block and Stokes-Adams attacks.

(2) *Ischæmic Heart Disease.* Wood and Wolferth (1934) first drew attention to the “huge” T wave inversion that sometimes occurred during cardiac infarction. Two examples of this are shown in Fig. 2. Note, however, that the feature of this disturbance is that the T waves are narrow, sharp, and symmetrical—the so-called “coronary type” of inversion. This phenomenon is encountered in “transmural” infarction (Fig. 2A) with loss of R waves, or more commonly in acute anterior ischæmia (Fig. 2B), or in subendocardial infarction.

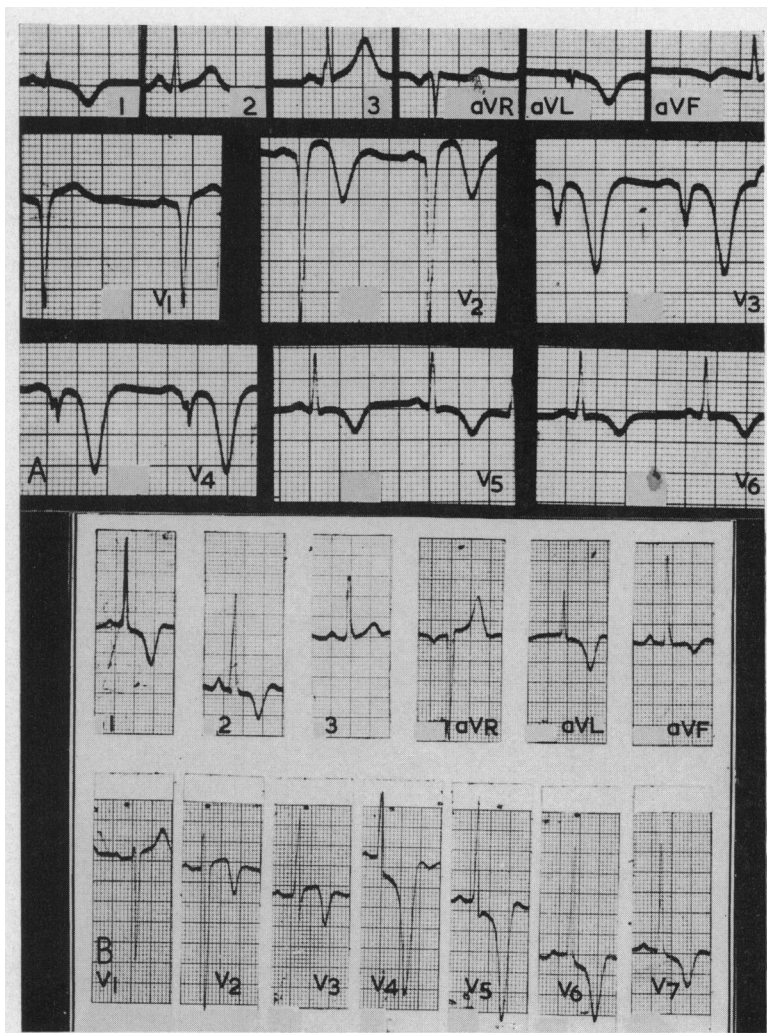


FIG. 2.—Symmetrical giant T wave inversion in 2 patients with cardiac infarction.

(3) *Bradycardia.* Scherf (1944) showed that in patients with myocardial disease, T wave inversion became more obvious after a long R-R interval, and he suggested that the larger diastolic load might affect repolarization. Szilagyi and Solomon (1959) confirmed the alteration in the T waves from beat to beat, dependent on the diastolic filling period. Their patient had complete heart block and Stokes-Adams attacks with massive T wave inversion, the largest T waves being associated with the longest R-R intervals. When partial heart block was restored some days after the syncopal attack, symmetrical T wave inversion appeared with the largest wave occurring after a blocked sinus beat. In

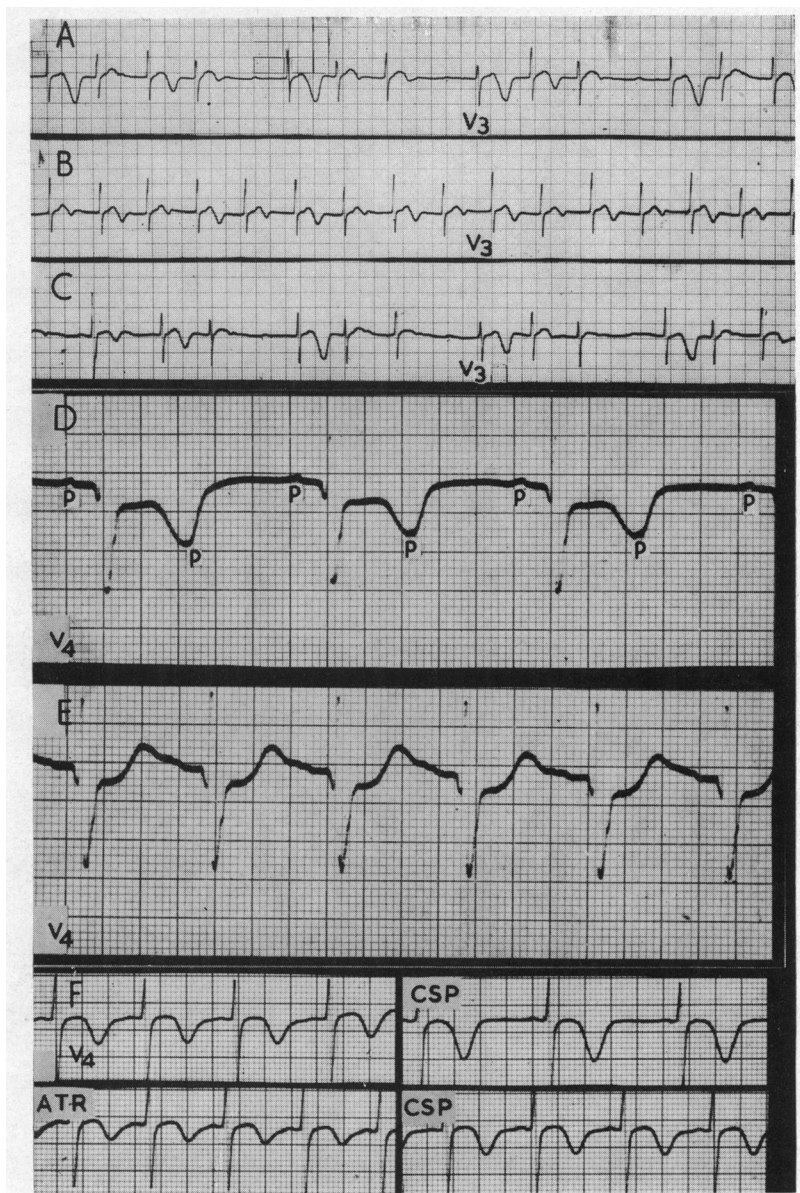


FIG. 3.—See text.

Fig. 3A, partial block was present. The largest negative T wave followed the longest R-R interval. After atropine (B), block disappeared, alternans was present, and the large T waves subsided, only to recur after carotid sinus pressure (C). The tracing came from a patient with a ruptured sinus of Valsalva aneurysm. Tracing (D) is recorded from a patient with ischæmic heart disease in whom 2:1 block spontaneously changed to sinus rhythm (E). There

were no symptoms at the time. Note the marked change in the T waves with the slowing of the ventricular rate. Tracing (F) was recorded from a patient with ischæmic heart disease, in whom the T wave configuration clearly altered when the heart rate was slowed by carotid sinus pressure. After atropine administration, the effect of carotid sinus pressure was still present but was less marked.

It is not clear whether the changes in T wave are

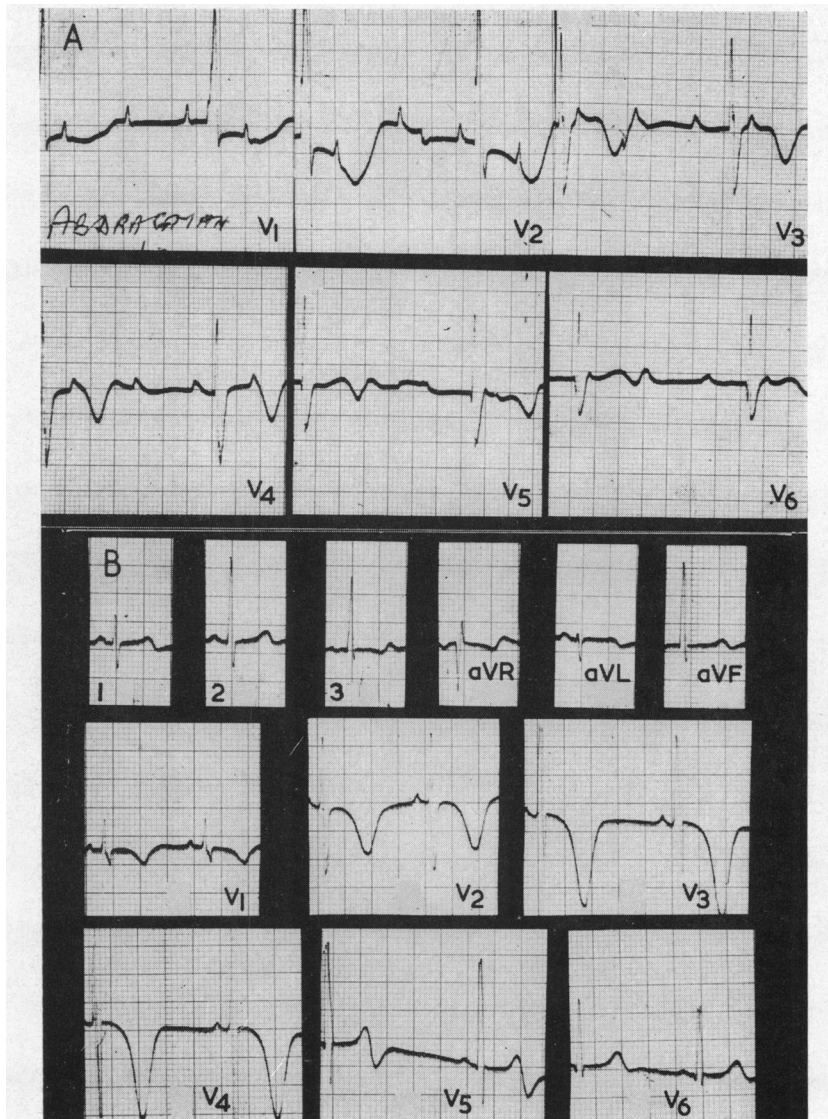


FIG. 4.—Giant T wave inversion in right ventricular hypertrophy.

entirely due to an alteration in heart rate, i.e. the effect of the prolonged diastolic filling period, or to a direct effect of the vagus nerve on the repolarization process.

(4) *Right Ventricular Strain.* Abnormalities in right depolarization may be involved in the production of giant negative T wave changes, encountered in conditions associated with enlargement of the right ventricle. Giant T waves can occur in severe right ventricular hypertrophy as is shown in Fig. 4. The first tracing (A) came from a child of

2 years with congenital heart block and idiopathic pulmonary hypertension, and the second from a boy of 14 with idiopathic familial primary pulmonary hypertension (both proven by cardiac catheterization and necropsy).

(5) *Metabolic Disturbances.* T and U wave fusion in hypokalaemia may create the impression of QT prolongation. However, giant deformity of the T waves does not occur. Aber and Jones (1965) have recently drawn attention to the effects of steroids in inducing giant T wave changes in complete heart block. In Fig. 5A the effects of a slow

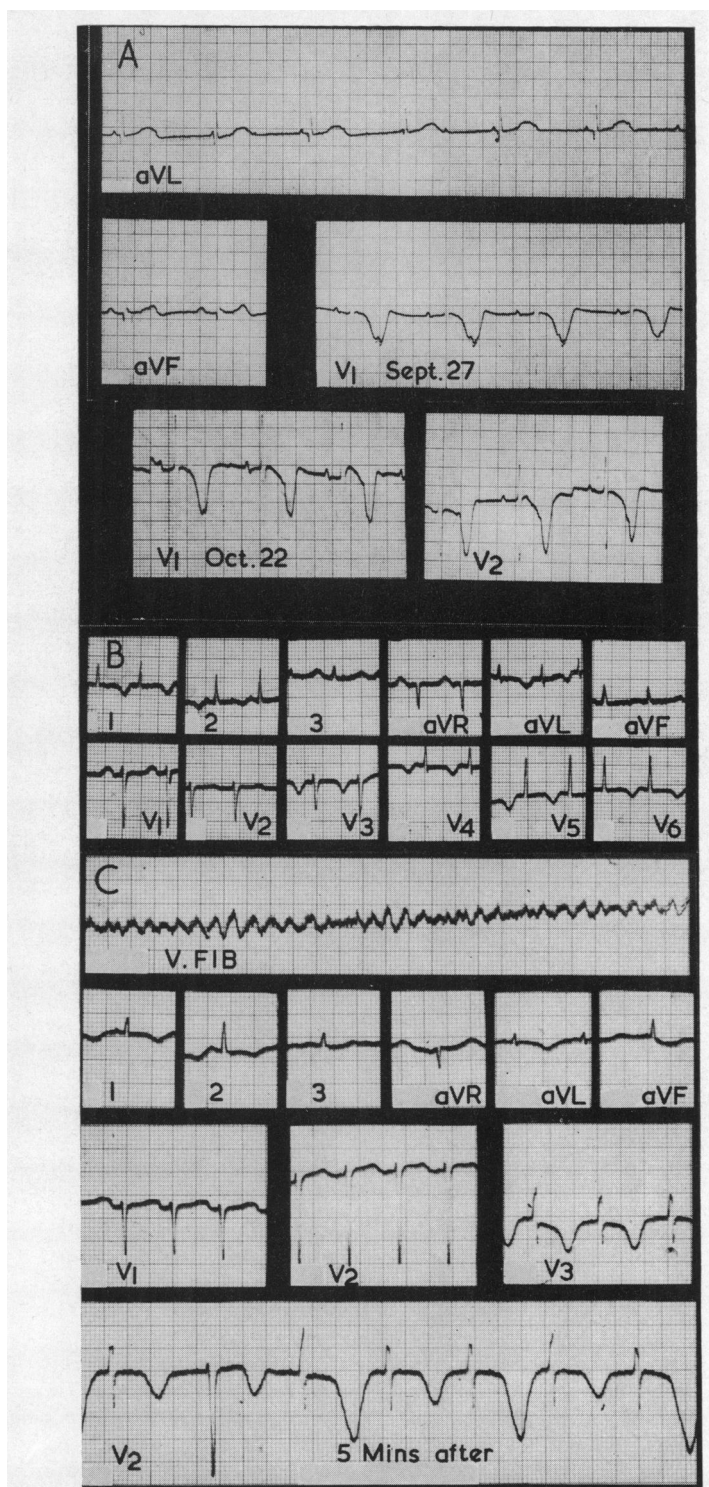


FIG. 5.—(A) Giant T wave inversion in a patient with congenital complete heart block and kwashiorkor. (B) Tracing from a patient with hypocalcemia. (C) After recovery from spontaneous ventricular fibrillation, alternans with marked T wave inversion is present.

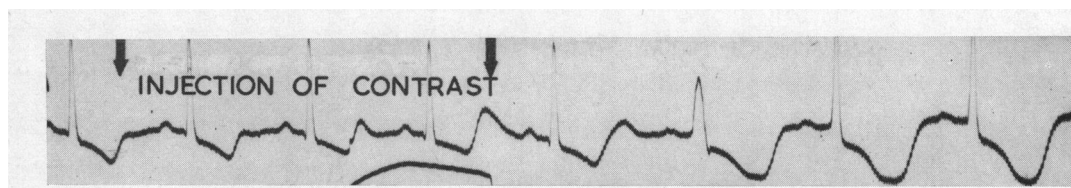


FIG. 6.—Changes in T wave contour during coronary arteriography.

heart rate plus metabolic disturbances are shown in a child aged 4 with congenital heart block and kwashiorkor. The sharp T wave inversion pattern as described by Smythe, Swanepoel, and Campbell (1962) is shown in an exaggerated form. The serum potassium was normal.

The classical metabolic cause of a long QT is hypocalcaemia (Fig. 5B). This young Bantu girl suffered from miliary tuberculosis with intestinal malabsorption and tetany, and she had a serum calcium level of 4.6 mg./100 ml. The QT was prolonged but the T wave was not abnormally enlarged. After an attack of spontaneous ventricular fibrillation, during which she lost consciousness, she was restored to sinus rhythm by external cardiac massage and electrical defibrillation. The tracing (Fig. 5C) then showed alternans, respiratory in origin, with gross giant negative T waves as the ventricular rate slowed. Necropsy showed no evidence of coronary vascular disease. The changes cannot be attributed to metabolic disturbance alone, but the effect of both cerebral and ionic changes acting together may well be responsible.

(6) *Coronary Angiography and Effect of Drugs.* We have observed deepening and expansion of the T waves soon after contrast material had been injected into a coronary artery (Fig. 6). This is transient, occurs fairly frequently, and is most unlike the changes associated naturally with coronary artery disease. According to Mason Sones (1965, personal communication), saline injections cause no change. Whatever the mechanism responsible for the temporary metabolic derangement of depolarization, there is quite a similarity between the changes produced during coronary angiography, and those occurring after a Stokes-Adams attack. Fox, Weaver, and March (1952) described a patient with Wolff-Parkinson-White syndrome, where intravenous procaine amide produced a transient bradycardia, 2:1 block, and massive negative T waves. Quinidine and procaine amide regularly deform the QT segment, but rarely to this degree.

(7) *Cerebral Causes.* Burch, Meyers, and Abildskov (1954) originally drew attention to the occur-

rence of profound T wave changes developing during cerebral disorders. Since then, a voluminous literature has developed based on clinical observations and experimental work (Hugenholtz, 1962; Hersch, 1961; Levine and White, 1962; Cropp and Manning, 1960; Koskelo, Punsar, and Sipilä, 1964; Hayashi *et al.*, 1961; Fentz and Gormsen, 1962; Wasserman *et al.*, 1956; Srivastava and Robson, 1964; Brink, 1960; Porter, Kamikawa, and Greenhoot, 1962; Náva, Marchetti, and Tartara, 1957; Melville *et al.*, 1963). The abnormal T waves resemble very closely those occurring after a Stokes-Adams attack. They have been encountered in patients suffering from subarachnoid haemorrhage, cerebral tumours, cerebral infarction, and after neurological procedures. They can be reproduced by hyperthalamic stimulation in animals, and abolished or prevented by nerve or cord section (Porter *et al.*, 1962; Náva *et al.*, 1957; Melville *et al.*, 1963). A typical example is shown in Fig. 7 from a 30-year-old Cape coloured woman admitted with a sudden left carotid artery occlusion. The electrocardiogram (A) was taken the day after cerebral angiography and had returned to normal (B) a day later.

SUMMARY

It is fair to conclude that massive T wave inversion can be produced by several causes. Symmetrical, deep, and narrow inversion of the T waves is usually due to ischaemic heart disease and ventricular hypertrophy, particularly that of the right ventricle. A special type of giant T wave has been noted following Stokes-Adams attacks associated with complete heart block. The T waves are deep, blunt, broad, often bizarre, with a prolonged QT_c and are usually maximal in leads V2 and V3. These changes are apparently particularly associated with syncope due to ventricular fibrillation. A slow heart rate with prolongation of ventricular diastolic filling time and ventricular distension may be an important component. However, cerebral disorders are probably the most important clinical cause of bizarre T wave distortion. It is likely that changes encountered in patients after Stokes-Adams attacks are basically cerebral in origin.

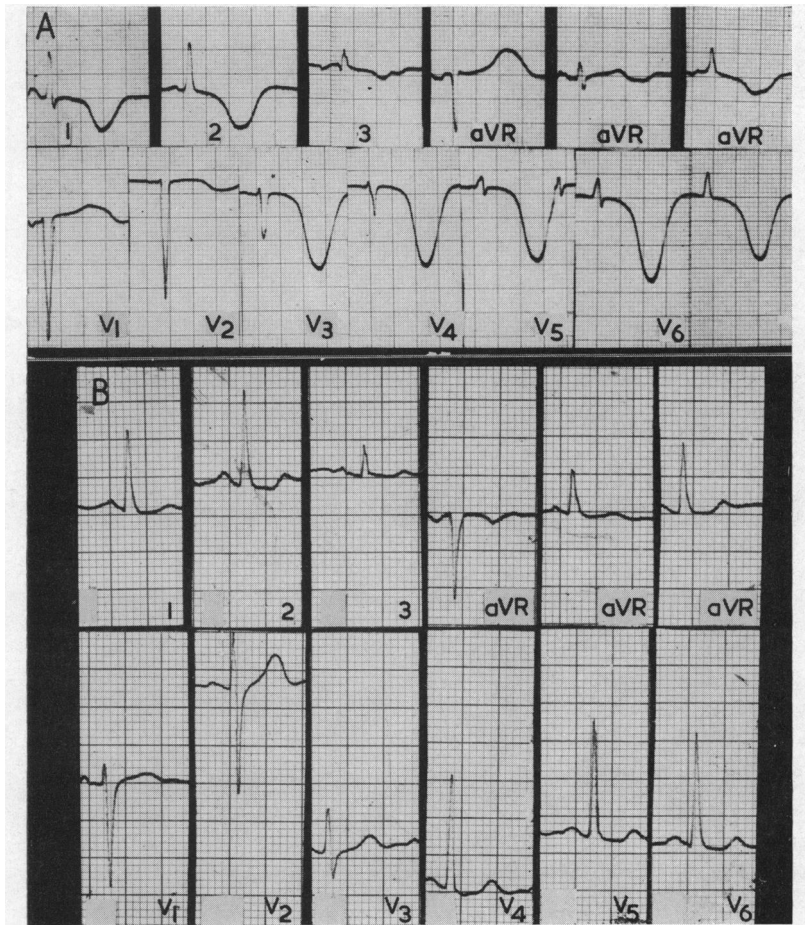


FIG. 7.—Tracings taken immediately after left carotid artery occlusion. (A) and (B) a day later.

We wish to thank the Medical Staff of Groote Schuur Hospital for freely referring their patients and the Superintendent, Dr. J. G. Burger, for permission to publish.

Our special thanks are due to the Council for Scientific and Industrial Research and the City Council of Cape Town for their continued financial support.

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